Chapter 2 : Literature Review

2.1 Nickel

Nickel is a silvery white and malleable metal. It has a high mechanical strength, is fairly hard and paramagnetic at ordinary temperatures. It oxidizes with difficulty when heated in air. Polished surfaces will, however, gradually corrode upon atmospheric exposure.

Nickel is slowly soluble in hydrochloric and in dilute sulphuric acids. Dilute nitric acid rapidly attacks nickel but immersion in concentrated sulphuric acid renders the surface of nickel "passive", which is not readily attacked. Nickel alloys were known and treasured by man for thousands of years before nickel was identified as a metallic element. The first commercial application of nickel was in the making of copper-nickel-zinc alloy, known as German silver or nickel silver, that began in the early nineteenth century. Nickel electroplating had come into commercial practice in the 1840's and by 1870 became one of the principal end uses of nickel (Nriagu, 1980).

2.1.1 Uses of Nickel

Nickel is found in many objects in our daily life. It is used in electroplating and in certain alloys as it renders strength and resistance to corrosion, the most common being the stainless steel. Nickel is found in diverse commodities such as automobiles, batteries, coins, costume jewellery, inks and varnishes, dyes, surgical implants, sinks and utensils. The major uses of refined nickel in industry include electroplating, alloy production and fabrication, the manufacture of nickel-cadmium batteries and electronic components, and the preparation of catalysts for hydrogenation of fats and methanation.

The waste generated from these industries contain nickel in various concentrations. In absence of proper treatment and disposal methods, nickel contamination in soil or water would occur and cause toxic exposure to living organisms (Waldron, 1980).

2.1.2 Nickel in Vegetation

Nickel is a non-essential trace element that occurs in trace amounts in most plants. If nickel exceeds threshold limit, inhibitory effects take place. The most critical factor in evaluating the impact of nickel on an ecosystem is the uptake

and accumulation of nickel in various organs of plants and animals. The mechanism of absorption of nickel by plants is influenced by the amount and form of nickel in the soil. It also impedes the plant growth which will affects the source of food for higher organisms, in relation to the entire food chain (Nriagu, 1980).

The transfer of nickel from the soil to the plant depends primarily on the following factors:

(1) Soil pH

The uptake of nickel is enhanced by the reduction in pH, especially below 6.5. This is caused by the breakdown of iron and manganese hydroxides which form stable complexes with nickel. The release of nickel from these sites facilitates the movement of nickel into the plant root and thereby accounts for higher levels of accumulation.

(2) Metallic interactions.

Metallic interaction has an important role on nickel uptake. Interactions among metals are mutual or reciprocal in nature and affect plant growth (Olsen, 1972).

(3) Concentration of nickel.

Plants accumulate nickel and other heavy metals depending on their availability in the soil.

The content of nickel in vegetation is usually below 1 μg/g on dry weight basis except where nickel rich substrates such as serpentinite are involved. For example, Vanselow (1966) reported 0.05 to 5 μg/g for field grown crops and natural vegetation. Conner et al. (1975) reported mean values in the range of 0.20 to 4.5 μg/g for nearly 2000 specimens of field crops and natural vegetation from United States. Although the concentration of nickel in vegetation growing on "normal" soils seldom exceeds 5 μg/g , this is not valid when ultrabasic substrates such as periodotite or serpentinite are involved. The nickel content in dried vegetation from serpentinite areas often is as high as 100 μg/g .

The discovery of Haselhoff (1893) on the toxicity of nickel on vegetation, generated research interest among scientists. The toxicity of nickel in plants is characterized by chlorosis of leaves, stunting of roots and deformation of various plant organs (Nriagu, 1980). The poisoning of plant enzymes is considered to be the most significant toxic action of this element (Bowen, 1960).

2.1.3 Distribution and Behaviour of Nickel in the Aquatic Environment

The behaviour of nickel in the aqueous environment is governed by its reaction with both soluble species and particulate material. Complexation is possible which forms soluble nickel-organic and nickel-inorganic species. Interaction with solid phases may occur by any of the following mechanisms:

1) Direct adsorption on fine grained inorganic particles such as clays.
2) Adsorption to, or co-precipitation with, hydrous ferric and manganic oxide which may then be sorbed to clay surfaces.
3) Complexation or adsorption with natural organic particles (e.g. detritus, algae, bacteria).
4) Ion-exchange with colloids.
5) Direct precipitation.

2.1.3.1 Nickel in Streams and Rivers

Several studies report the observed concentration of nickel in flowing river water (Nriagu, 1980). Table 2.1 shows data for Malaysian rivers, reported by The Department of Environment, Malaysia (1995).

Table 2.1: Nickel in river water- 1995.

State	River name	Nickel (mg / L)
Terengganu	Kemaman	0.9000
Johor	Benut	0.1930
Sabah	Putatan/Moyong	0.0762
Johor	Sedeli Kechil	0.0650
Johor	Sedeli Besar	0.0500
Sarawak	Miri/Lutong	0.0500
Sabah	Labok	0.0400
Sabah	Sugut	0.0395
Johor	Batu Pahat	0.0317
Johor	Tukang Batu	0.0310
Sabah	Kinabatangan	0.0250
Johor	Endau	0.0220
Johor	Johor	0.0182
Sabah	Silabukan	0.0100
Sabah	Mengalong	0.0100
Sabah	Umas-Umas	0.0100

(DOE, 1995)

In the major rivers of the world, the forms of nickel were estimated as follows (Gibbs, 1973, 1977):

Table 2.2 : Forms of nickel in the major rivers of the world

Form	Percentage %
dissolved	0.5
adsorbed	3.1
precipitated coating	47.0
organic matter	14.9
crystalline material	34.4

(Gibbs, 1973, 1977)

2.1.3.2 Nickel in Lakes

The concentration of nickel in lakes is reflected by the nickel contents of the surrounding bedrock unless modified within the lake. The modification of concentration occurs in lakes due to the following reasons :

i) Detritus materials become incorporated in lake sediments and undergo diagnoses

ii) Discharge of industrial/municipal wastes containing heavy metals.

Influents having a nickel concentration of 1 to 3 μg/L is not considered to be a serious source of pollution for lake waters.

2.1.3.3 Nickel in Estuaries

In estuaries, the higher concentrations are caused by association of nickel with fine grained particles, organic matters, iron and manganese hydroxide. Trefry and Presley (1976) found that nickel in suspended matter in river waters does not vary much from that in suspended matter in estuary. This similarity argues against any significant desorption of nickel from the suspended matter, as the sediment changes. However, nickel in the delta sediments is significantly lower than in the suspended matter which shows that desorption or postdesorptional migration of nickel occurs.

2.1.3.4 Nickel in Wastewater

Major content of nickel in water bodies comes from industrial and municipal wastewater. Table 2.3, shows data from a study by Klein *et al.* (1974) on New York City wastewater collection.

Table 2.3 : Nickel in various wastewater

Sources	Percentage of Nickel (%)
Electroplaters	62.0
Other industrial	3.2
Runoff	9.8
Residential	25.0

(Klein *et al.*, 1974)

Table 2.3 shows that electroplating plants are the main source of nickel in wastewater and residential sources contribute 25 % of the total nickel in wastewater. Another survey by Teh (1989), involving 54 metal finishing premises located in the Klang Valley, Malaysia showed that the concentration of nickel was 94.0 mg/L. This value by far exceeded the Standard B of Environmental Quality (Sewage and Industrial Effluent) Regulation 1979 discharge limits of Malaysia which is 1.0 mg/L (Appendix 1).

2.1.4 Toxicity of Nickel

2.1.4.1 Effect of Nickel on Marine Organisms

Nickel occurs in aquatic environment mainly in the form of divalent cation.

Nickel enters the marine environment both as a result of natural mineralisation and anthropogenic factors. Usually the level of nickel in seawater from locations unaffected by mineralisation or pollution is less than 1.0 μg/L.

Marine Fauna

Table 2.4 shows the acute toxicity of marine fauna, 12 invertebrate species and 2 vertebrate (fish) species are presented. The toxicity of nickel presented as 96-hour (LC50) acute values (WRC, 1984), is as follows.

Table 2.4: Acute toxicity (96-h LC50) of Nickel (as chloride) to marine fauna

Species	LC50 (µg/L)
ANNELIDA	
Ctenoderilus serratus	17000
Neanthes arenaceodentata	49000
Nereis virens	25000
MOLLUSCA	
Nassarius obsoletus	72000
Mya arenaria	320000
Mercinaria mercinaria	320
CRUSTACEA	
Acartia clausi	2080
Nitocra spinipes	600
Heteromysis formosa	152
Mysidopsis bigelowi	634
Mysidopsis bahia	508
ECINODERMATA	
Asterias forbesi	150000
FISH	
Fundulus heteroclitus	350000
Menidia menidia	7960

(WRC, 1984)

A study by Timourian and Watchmaker (1972) showed that the lowest nickel concentration required to produce an adverse effect in a marine animal was 58 µg/L. It was found that this concentration delayed development of embryos of the sea urchin, *Lytechinus pictus.*

Marine Flora

In a study by Clendenning and North (1960), nickel concentration of 2000 µg/L caused 50% inactivation of photosynthesis in the macroalgae *Macrocystis pyrifera* (brown algae). Skaar et al. (1974) reported that a nickel concentration of 1000 µg/L would reduce the growth of *Phaeodactylum tricornutum* (phytoplankton). In another study on mixed culture of phytoplankton, Hollibaugh et al.(1980) observed growth inhibition at concentration of 581 µg/L.

2.1.4.2 Effect of Nickel on Freshwater Organisms

Acute Toxicity of Fish

The toxicity of nickel for fresh water organisms depends on animal species, water hardness, pH and other conditions (Pickering and Henderson, 1966). A 96-

hour median lethal concentrations (96-h LC) of nickel from 2.5 mg Ni/L to 110 mg/L were reported for various species of fish. Among the different species of fish, rainbow trout was apparently one of the most resistant species. A study by US Environment Protection Agency in 1980 showed some adverse effects on two species of fresh water fish; rainbow trout and fathead minnow. The adverse effect can be caused by extended periods of exposure to nickel concentrations greater than 0.4 mg/L to 0.5 mg/L in hardwaters. Therefore it is recommended that for the protection of fish in such waters (250 mg/L as $CaCO_3$) the average concentration of nickel should not exceed 200 µg/L . In soft waters (44 mg/L as $CaCO_3$) embryoes and larvae of fathead minnow were adversely affected at 0.11 mg Ni/L and therefore in such waters a maximum average concentration of 50 µg Ni/L is recommended.

Toxicity in Freshwater Invertebrates

The reproduction in *Daphnia magna* in soft water (hardness 45 mg/L as $CaCO_3$) was impaired by 16% over an exposure period of 21 days (Biesinger and Christensen, 1972). *Daphnia magna* indicated acute LC 50 to be larger with a greater degree of water hardness (EPA,1980). In another study , Buikema et al. (1974) gave a probit-derived 96- hr LC50 of 2.6 mg Ni/L to the rotifer *Philodina acuticornis*.

Toxicity in Freshwater Plants

Adverse effects of nickel in algae was found with the concentrations approximately above 50 μg/L (Spencer and Green, 1981). However, a study on *Scenedesmus* sp. drew attention because the species was not affected by a nickel concentration of 3.0 mg/L and categorized as metal tolerant strain (WRC, 1984). The data for *Lemna*, a higher flowering plant also showed that the level proposed for the protection of algae could be adequate for the protection of higher plants (WRC, 1984).

2.1.5 The Effect of Nickel on Human Health

Since nickel is found in soil and water. It is available in natural food and water. Nickel is found in vegetables, fruits and grains. The nickel content of plants depends on the quantity in the soil in which they grow and the ability of a plant to incorporate nickel.

There are two significant routes of entry of nickel into the human body, inhalation and ingestion. The inhaled nickel may be transported into the blood and excreted, or it may be retained in the lung. In a study on nickel content in blood plasma among workers in a nickel refinery, Hogetveit and Barton (1976)

found 0.74 μg/100 mL, 0.6 μg/100 mL and 0.42 μg/100 mL among those who were working in electrolysis, furnace and control sections respectively. Solubility is the primary factor which determines the availability of nickel in the lungs. Nickel oxide or metallic nickel, which is insoluble in body fluids, may be retained for a long period.

Nickel is most often found as Ni^{2+} and it can form co-ordination complexes with organic molecules. Complexes with amino acids through binding with carbonyl and alpha amino acid groups are common in nickel and it has high affinity for sulphur (National Academy of Sciences, 1975). Among nickel compounds, the most acutely toxic to man is nickel carbonyl. According to Sunderman (1970) and Vuopala et al. (1970), clinical symptoms of acute nickel carbonyl poisoning include frontal headache, nausea, vomiting, insomnia and irritability. Persistent symptoms include, constrictive chest pains, dry cough, hyperpnea, cyanosis, occasional gastrointestinal disturbances, sweating, visual disturbances and severe weakness.

Other nickel forms also cause adverse respiratory effects on man. Inhalation of nickel sulphate aerosols, has been found to cause chronic rhinitis and nasal sinusitis in Russian nickel platers (Nriagu, 1980). Many authors have reported nickel as a carcinogenic metal. Table 2.5 shows a possible relation between

exposure to nickel and the incidence of cancer (Nriagu, 1980).

Table 2.5: Possible relation of cancer and nickel compounds

Compounds	Population	Nature of tumor
Ni carbonyl, $Ni_3 S_2$ dust, nickel copper oxide	Nickel refinery	Nasal sinus cancer, lung cancer, other neoplasma
Nickel ore dust	Nickel refinery	Nasal cavities, larynx, lungs
$Ni_3 S_2$, NiO, $Ni_2 O_3$, $Ni(CO)_4$, $NiSO_4$, $NiCl_2$	Nickel refinery	Pulmonary and nasal cavity cancers
Nickel carbonyl, nickel ore dust	Nickel mining, nickel refinery	Bronchii and upper respiratory
Nickel	Nickel refinery, metal workers	Squamous cell and oat cell carcinoma
Nickel carbonyl, nickel oxide, nickel subsulfide	Nickel refinery	Nasal cavity and lung cancer

(Nriagu, 1980)

It has been found that nickel also may cause dermatitis. "Nickel itch", a disease among workers handling nickel salts was once common, especially in hot or humid environments. Nickel was the most common allergen tested by the North American Contact Dermatitis Group (1973). A study showed that, 11% in a group of 1200 persons had positive patch test for nickel.

2.1.6 Recommended Environmental Quality Standards (EQS)

Environmental quality standards recommended by Water Research Centre (WRC, 1984), are given in Table 2.6. The values given are based on chemistry and behaviour of nickel in the environment and effects of nickel on human, fish, other biota, agricultural crops etc.

Table 2.6 : Environmental standard quality by WRC, 1984

Use	Average concentration (μg/L)
Fresh water	
Direct abstraction to potable supply	50*
Protection of fresh water fish	
Total hardness less than 50	50
as mg $CaCO_3$ / l 50 - 100	100
100 - 200	150
greater than 200	200
Protection of other freshwater life and associated non- aquatic organisms	
Total hardness less than 50	8
as mg $CaCO_3$ / l 50 - 100	20
100 - 200	50
greater than 200	100
Irrigation of crops	150
Abstraction for food processing industry	50*
Bathing and contact water sports	500
Saltwater	
Protection of saltwater fish and shellfish	30
Protection of other saltwater life and associated non-aquatic organisms	30
Bathing and contact	500

* indicates mandatory values. (WRC, 1984).

2.2 Chromium

Earth's crust has been estimated as containing around 200 mg/kg of chromium. In natural deposition, chromium is found mainly in trivalent state. Naturally occurring hexavalent chromium has also been found. Chromium is present mainly in spinel-type minerals such as iron and magnesium chromo-aluminates, and the most important one is chromite ($FeO.Cr_2O_3$). Chromite is the only commercial source of chromium ore used for refining industry. Chromite ores are mostly mined in the former USSR, South Africa, Albania, Zimbabwe and Philippines with annual production of chromium from chromite, reaching 10^7 tonnes (Papp,1988). Out of this about 60 - 70 % was used in alloys (Stern, 1982) including stainless steel which contains Fe, Cr and Ni in varying proportions according to the properties required. Alloy steels usually contain about 10 - 26 % Cr.

The refractory properties of Cr are exploited in the production of refractory bricks for lining furnaces and kilns, which accounts for approximately 15 % of the chromate ore used. General chemical industries producing chrome alum [Cr(III)] for tanning leather, pigments and wood preservatives (sodium dichromate) consume about 15 % of the chromium produced. About 4 % is converted into chromic acid and used for electroplating or as an oxidant. Table

2.7 shows the major uses of chromium in producing chemicals (McGrath, 1995).

Table 2.7 : Some uses of chemicals containing Cr

Antifouling pigments	Metal finishing
Antiknock compounds	Metal primers
Catalysts	Mordant
Ceramics	Phosphate coatings
Corrosion inhibitors	Photosensitisation
Drilling mud	Pyrotechnics
Electronics	Refractories
Emulsion hardeners	Tanning
Flexible printing	Textile preservatives
Fungicides	Textile printing and dyeing
Gas absorbers	Wash primers
High temperature batteries	Wood Preservatives
Magnetic tapes	

(McGrath, 1995)

2.2.1 Chromium in Vegetation

Concentrations of Cr in plants are extremely low due to the lack of plant-available form in most soil. Cr which is present in soil in mostly the trivalent state is adsorbed on other metal hydroxides and is not available to plants (Cary,1982). The background concentration in plants is 0.23 mg/kg on an average, and in general, concentrations are less than 1 mg/kg (Bowen, 1979). Various authors conducted studies to observe Cr uptake in plants by adding various Cr compound to soils. It was shown that recovery of added Cr by plants is extremely inefficient. Cary et al.(1977) added up to 1 % chromium as $Cr(OH)_3$ and grew wheat (*Triticum aestivum*), alfalfa (*Medicago sativa*) and buckwheat (*Fagopyrum esculentum*). He observed the Cr concentration in wheat grain did not increase but there was higher accumulation in the wheat leaves and stems with increasing amount of Cr in the soil. Alfalfa and buckwheat also showed some increases in chromium concentration. In solution culture of rice plants (*Oryza sativa*), Cr(III) and Cr(IV) behaved in the same way. 95 % Cr was retained in the root with less than 1 % was translocated to the leaf (Myttenaere and Mousny, 1974).

2.2.2 Distribution and Behaviour of Cr in Aquatic Environment

Chromium occurs in natural water bodies as a result of mineral weathering, sediment load and precipitation. Chromium is also present as a result of industrial pollution.

2.2.2.1 Chromium in Streams and Rivers

Chromium is present in both soluble and insoluble forms in river water. Most of the soluble chromium is present as hexavalent chromium which only contributes a few percent of the total. Chromium (III) is mostly present in suspended solids as organics, minerals and occluded oxides. Gibbs (1977) reported the solution phase concentration in the Amazon River and in the Yukon River as 2.0 and 2.3 μg/L respectively. These two rivers are considered to represent unpolluted systems draining watersheds with a wide variety of mineral rocks. In another study, by National Research Council of Canada (1976), it was found that the concentration of chromium in 95.9 % of the samples of stream and river water in Canada is less than 10 μg/L . Table 2.8 shows chromium level for Malaysian rivers reported by The Department of Environment, Malaysia (1995).

Table 2.8 : Chromium in river water - 1995

State	River	Chromium (mg/L)
Kedah	Merbok	0.8000
Sabah	Kalabakan	0.2000
Sabah	Silabukan	0.2000
Sabah	Kaya	0.2000
Sabah	Kinabatangan	0.2000
Sabah	Segama	0.2000
Sabah	Kalumpang	0.2000
Sabah	Tawau	0.2000
Sabah	Umas-Umas	0.2000
Sabah	Putatan	0.1480
Johor	Endau	0.1260
Sabah	Labok	0.0950
Sabah	Membakut	0.0500
Sabah	Papar	0.0500
Pahang	Mentiga	0.0500

(DOE, 1995)

2.2.2.2 Chromium in Wastewater

Chromium in water bodies mainly comes from industrial and domestic wastewater. According to Yapijakis and Papamichael (1987), industrial sources contribute 68 % of chromium in the influent to sewer in New York, USA. Chromium containing effluents are released by following activities : metal plating, anodizing, ink manufacture, dyes, pigments, glass, ceramics, glues, tanning, wood preserving, textiles and corrosion inhibitors in cooling water. Both Cr(III) and Cr (VI) can be present in these effluents. Cr(VI) however predominates in raw effluent from plating works (Beszedits, 1988). A survey by Teh (1989), involving 54 metal finishing premises located in the Klang Valley (Malaysia), showed that the concentration of Cr(VI) was 6.6 mg/L and 94 mg/L for Cr(III). These values exceeded the Standard B of Environmental Quality (Sewage and Industrial Effluent) Regulation 1979 discharge limits of Malaysia which is 0.05 mg/L and 1.0 mg/L respectively (Appendix 1).

2.2.3 Toxicity of Chromium

2.2.3.1 Effect of Chromium on Marine Organisms

Hexavalent Cr compounds are more soluble and relatively stable in marine

waters than trivalent forms which are less soluble and readily form complexes with both organic and inorganic materials. Natural level of chromium in oceanic waters is approximately 0.04 μg/L .

Marine Fauna

Table 2.9 shows the acute toxicity (LC50) of hexavalent chromium Cr on marine fauna. The 96-h LC50 values show considerable interphyletic variation. The most sensitive phylum appears to be the annelids (WRC, 1984).

Table 2.9 : Acute toxicity (LC50) for hexavalent chromium

Phylum / species	Chemical form	Duration of exposure	LC50 (µg / L)
Annelids			
Capitella capitata	Chromium trioxide	96 h	5000
Neanthes arenaceodentata	Potassium dichromate	96 h	3100
Ctenodrilus serratus	Chromium trioxide	96 h	4300
Nereis virens	Potassium chromate	96 h	2000
Crustaceans			
Acartia clausi	Potassium dichromate	96 h	6600
Tigriopus japonicus	Potassium dichromate	96 h	17200
Mysidopsis bigelowi	Potassium dichromate	96 h	4400
Fish			
Fundulus heteroclitus	Potassium chromate	96 h	91000
Menidia menidia	Potassium dichromate	96 h	20100

(WRC, 1984)

Toxicity data for trivalent and marine fauna are somewhat limited. Table 2.10 shows acute toxicity values for one species of mollusc and one species of crustacean (WRC, 1984).

Table 2.10 : Acute toxicity (96-h LC50) for trivalent chromium

Phylum/species	Chemical form	LC50 (mg / L)
Mollusc		
Crassostrea virginica	Chromic chloride	10.3
Crustacean		
Sosarma haomatochoir	Chromic chloride	56.0

(WRC, 1984)

Marine Flora

Information on the effects of chromium on marine flora is limited to the inhibition of photosynthesis in a single species, the giant kelp *Macrocystis pyrifera* . It has been reported 50 % inhibition of photosynthesis in a four-day exposure to 5000 µg/L of chromium and 10 to 20 % inhibition in a five days exposure to 1000 µg/L of chromium (WRC, 1984). Holibaugh et. al (1980) studied the effect of hexavalent chromium on the marine phytoplankton

Thalassiosira aestevalis. It was found that, inhibition of growth took place when the chromium concentration exceeded 1000 nM (about 52 μg/L). Study on marine microflora by Aubert et. al (1975) demonstrated that the growths of phytoplankton *Asterionella japonica* and *Diogenes* spp. were inhibited at chromium levels of 40,000 μg/L, and 3200 to 6400 μg/L respectively.

2.2.3.2 Effect of Chromium on Freshwater Organisms

The movement of Cr (VI) across biological membranes is easier than Cr (III) unless its net charge is decreased by complexation. Cr (III) is described as more toxic (in terms of 96-h median lethal concentration) than Cr (VI) and the toxicity of Cr (III) is greater in softwater (EPA, 1980). Chromic salts and dichromates show more toxic effect to fish than chromates (WRC, 1984).

Toxicity to Fish

Table 2.11 shows the acute toxicity of hexavalent chromium to freshwater fish in terms of 96-h LC 50. The toxicity of chromium not only depends on the particular species involved, but also shows greater effect in soft water at low pH and high temperatures.

Table 2.11: Acute toxicity of hexavalent chromium to freshwater fish

Species	Conditions	96-h LC50 (mg/L)
Rainbow trout	mean weight 25 g pH 7.8	65.5
	pH 6.5	20.2
	Soft water	32
	Hard water	69
Silver salmon	Soft water	> 100
Fathead minnow	10 ° C	52
	25 ° C	37
Bluegill	Soft water	113
	Hard water	133

(WRC, 1984)

Trivalent chromium also shows a greater effect with water softness. Soft water enhances toxicity as compared with hard water. Table 2.12 shows the acute toxicity of trivalent chromium to fresh water fish.

Table 2.12 : Acute toxicity of trivalent chromium to fresh water fish

Species	Conditions	96-h (LC50) mg/L
Fathead minnow	Soft water	5.07
	Hard water	67.4
Bluegill	Soft water	7.46
	Hard water	71.9
Goldfish	Soft water	4.10
Guppy	Soft water	3.33

(WRC, 1984)

Toxicity to Invertebrates

Data on fresh water invertebrates indicate that under conditions of short term exposure to hexavalent chromium in most cases, a minimum concentration between 4 mg/L and 60 mg/L is required to cause harm. The acute toxicity of trivalent chromium appears to be similar to hexavalent chromium for concentrations ranging from 2 mg/L to 64 mg/L to harm a variety of invertebrates. Table 2.13 shows the acute values (96-h LC 50) for chromium (EPA, 1980).

Table 2.13 : Acute values (96-h LC50) for chromium

Species	Hardness (mg/l as $CaCO_3$)	LC50 (µg/L)
Hexavalent chromium		
Philodina acuticornis	25	3 100
	81	15 000
Physa heterostropha	45	17 300
	171	31 600 - 40 600
Gammarus pseudolimnaeus	45	67
Trivalent chromium		
Nais sp	50	9 300
Amnicola sp.	50	8 400
Daphnia magna	48	2 000
	99	27 400

(EPA, 1980)

Toxicity to Plants

The effect of chromium on aquatic plants is shown in Table 2.14. Hexavalent chromium as low as 10 µg/L reduced growth of the algae *Chlamydomonas*. The higher plant, water millfoil had root growth inhibited by 50 % by hexavalent chromium at 1.9 mg/L and trivalent chromium at 9.9 mg/L.

Table 2.14 : Plant values for chromium (EPA, 1980)

Species	Effect	Chromium concentration (μg/L)
Hexavalent chromium		
Navicula sominulum	50 % growth reduction	187 - 308
Chlamydomonas reinhardi	Reduction in growth	10
Myriophyllum spicatum (Eurasian watermillfoil)	50 % root weight inhibition	1900
Trivalent chromium		
Myriophyllum spicatum	50 % root weight inhibition	9900

(EPA, 1980)

2.2.4 The Effect of Chromium on Human Health

There are three different routes of entry for chromium into the human body. Gastro-intestinal route is the most important in physiological conditions, while in occupational exposure the airways are more important route of entry and uptake. In pathological conditions, uptake through skin is more significant.

Valency state of chromium compounds, water solubility, acidity of gastric juice and the passage time through the tract are the factors which control the uptake of chromium in GI-tract. Uptake in the airways is influenced by the particle size distribution of the inhaled aerosol and on the factors which govern the clearance time from the lungs (Langard, 1980).

Ingestion of more than 3 g of chromic acid is considered as lethal for an adult human being. It caused vomiting immediately after ingestion which persisted for 24 hours, followed by diarrhoea which persisted for 3 days (Langard, 1980). Haemorrhagic diathesis, epistaxis and haemorrhages were observed after I week. Clinical signs shows Necrosis of renal tubulary and liver occuring on the second day itself. Fatal cases of chromic poisoning have also been reported . Death followed 1-9 days after chromate ingestion and the patients suffered from convulsions during their final stages of illness. Inhaled aerosols which transport chromium containing compounds of low water solubility are deposited in the airways. Particles larger than 5 μm MMAD (Mass Median Aerodynamic Diameter) are deposited in the upper airways, while particles between 5-2 μm (MMAD) and those below 0.5 μm are deposited mainly in the bronchial tree and alveolus region respectively (Lippmann et al., 1980). Bronchial asthma due to inhalation of chromate dust or chromic acid fumes has been reported (Williams, 1969). Chromium compound also may cause damage to the skin. The effect of

chromium on the skin are of two types; primary irritant reactions, that is chrome ulcers and allergic contact dermatitis. The former causes corrosive reactions from certain chromium compounds which ultimately causes ulcers, while allergic contact dermatitis is mainly of occupational origin, which causes weeping eczema and spreads to areas other than the original contact points (Pedersen, 1982).

Carcinogenic Effects

Chromium compounds are also known for their carcinogenic effect especially when long term exposure is involved. High incidence of cancer in the respiratory organs have been reported among chromate workers. Inhalation of chromium in the form of dichromates also shows carcinogenic effect in human. In 1976, a study on workers of a chromate pigment plant by Equitable Environmental Health, in USA showed an excess risk of respiratory cancer. In another study on workers from die casting and electroplating plant, Silverstein et al. (1980) found an approximately twofold proportional excess of deaths due to cancer of the respiratory system.

2.2.5 Recommended Environmental Quality Standards (EQS)

Environmental quality standards recommended by Water Research Centre (WRC, 1984) are given in Table 2.15. The values given here are based on chemistry and behaviour of chromium in the environment and its effect on human, crops, fish and other biota .

Table 2.15 : Environmental standard quality by WRC, 1984

Use			Average concentration (μg/L)
Fresh water			
Direct abstraction to potable supply			50*
Protection of fresh water fish			
Salmonid fish	less than 50		5
Total hardness	50 - 100	mg $CaCO_3$ / L	10
	100 - 200		20
	greater than 200		50
Coarse fish	less than 50		150
Total hardness	50 - 100	mg $CaCO_3$ / L	175
	100 - 200		200
	greater than 200		250
Irrigation of crops			2000
Abstraction for food processing industry			50
Bathing and contact water sports			500
Saltwater			
Protection of saltwater fish and shellfish			15
Protection of other saltwater life and associated non-aquatic organisms			15
Bathing and contact			500

* indicates mandatory values. (WRC, 1984).